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THE CLINICAL SIGNIFICANCE OF THE RAMIFICATION OF THE RECURRENT LARYNGEAL NERVES.*

A Critical Anatomical Study.

H. PICHLER, M.D., and A. GISEL, M.D.,

Vienna, Austria.

Paralyses of the vocal cords occurring after strumectomies and other operations of the throat, or after traumas of the neck, present a number of questions which as yet have not been satisfactorily answered, either anatomically or physiologically. In the past numerous investigators have tried to explain the various behaviors of the vocal cords after partial or total paralysis of the recurrent nerve; however, the literature has shown wide differences of opinion.

In the course of recent years American authors have taken up this subject again, thus reviving discussion of an old problem. On the basis of more recent anatomical studies of the distribution of the recurrent laryngeal nerves, they called attention to a fact which, although generally known, obviously had not been given due consideration to date: the fact that the recurrent nerve sometimes divides before it enters the larynx.

In 1942 Weeks and Hinton pointed out extralaryngeal ramification of the recurrent nerve and the possible clinical significance of this condition in cases of paralyzed vocal

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cords. In a study on 28 cadavers these authors reported extralaryngeal division in 78 per cent.

In 1948 King and Gregg investigated the same subject, going a little further. Introducing their conclusions they attempted to explain the various and heretofore unexplained behaviors of paralyzed vocal cords on the basis of high or low division of the recurrent nerve, inside or outside of the zone which might possibly be damaged through surgery: "The type of injury accounts for the different positions which the paralyzed cords may occupy."

Quoting Piersol in his "Human Anatomy", Vol. II, King and Gregg state that the recurrent nerve divides into two main trunks: one to supply the abductor muscles; the other the adductors of the vocal cords. They point out that "the usual point of division is at, or near, the lower border of the cricoid cartilage on its posterior surface behind and mesial to the interior cornu of the thyroid cartilage;" however, it was observed by them and others that division of the nerve sometimes took place "at a lower level". No significance heretofore had been attributed to this anatomical finding, yet the authors stress the fact that the extralaryngeal point of bifurcation "offers the only rational explanation of all lower motor neuron vocal cord paralyses."

In 25 to 36.4 per cent of their material, *viz.*, in 8 among 32 dissected nerves and in 4 among 11 dissections, the division of the nerve occurred in what the authors designate as the "nerve injury zone", namely, "that portion of the thyroid space in which the nerve, by its location, is subject to injury during thyroid surgery." More details on this paper will be given later.

In 1951 Armstrong and Hinton studied 100 prepared recurrent nerves, in which they observed extralaryngeal division in 73 per cent. Most often the main trunk divided into two branches, but three, four and even five branches could be noted. In the majority of cases the site of ramification was in close relation to the inferior thyroid artery, or was present in the upper third of the extralaryngeal course of the nerve.

In 1952 Rustad and Morrison, studying 100 cadavers, concluded that branching may be present at any site, "starting from the level of the clavicula to the cricothyroid articulation." They also found that the course of the recurrent nerve on the right and left side was seldom the same. The results of these authors conform to King's opinion.

By permission of the authors, the following illustration has been reproduced from the paper presented by Rustad and Morrison (see Fig. 1).

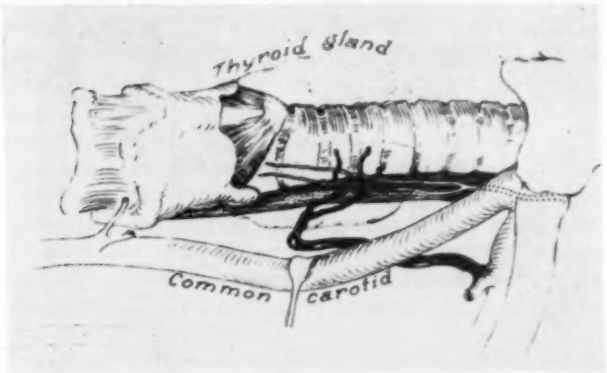


Fig. 1. Divided right recurrent laryngeal nerve. (Rustad and Morrison, Fig. 1, p. 241).

Likewise in 1952 the Belgian investigator Kluyskens reexamined King's findings on a larger scale, reporting on 150 cases (300 dissected nerves). He found high bilateral bifurcation in 127 cases; low bilateral bifurcation in 12 cases. In 11 cases low bifurcation was present at the right side; high bifurcation at the left; hence, the rate of extralaryngeal divisions figured 11.6 per cent. Kluyskens never observed unilateral low bifurcation at the left side. Summing up the anatomical evidence gathered in this study, Kluyskens stated that, on the whole, he confirmed King's results; in fact, he expressed the opinion that "low bifurcation occurs frequently enough to explain, on these grounds, all the various types

of paralysis occurring in thyroidectomies after traumatic lesions of the inferior laryngeal nerve."

In 1953, in Zurich, Clerf presented his view of the problem of paralyzed vocal cords after injury to a part or the entire nerve supply of the intrinsic muscles of the larynx. According to Clerf, the various behaviors of the paralyzed cords, heretofore "a source of much confusion," have been satisfactorily explained both anatomically and clinically through the works of King and Gregg, Morrison, et al. "While their explanation has been offered recently, their solution appeared in an excellent anatomical plate in Toldt's Atlas of Human Anatomy, published in 1904. In this is shown an extralaryngeal division of the recurrent nerve into anterior and posterior branches distributed to the abductor and adductor muscles respectively." Following King's rationale, Clerf believes that "with interruption of the entire recurrent nerve neither abductor nor adductor muscles function, and the corresponding cord assumes an intermediate position with loss of tension. With injury to one of the subdivisions of the recurrent nerve occurring extralaryngeally, the position assumed by the vocal cord depends upon whether the branch to the adductor or abductor group has been injured."

We readily admit that we, too, were at first much impressed by King and Gregg's anatomical reason for the *vagaries* in the behaviors of paralyzed vocal cords. The essential points of their very plausible explanation can best be demonstrated in the illustrations below (see Figs. 2 and 3).

Obviously, the essentially new feature of King-Gregg's explanation is the clinical significance attributed to the presence of an extralaryngeal motor branch of the recurrent nerve; *viz.*, the extralaryngeal posterior ramus supplying the abductor muscle. If the posterior ramus branches off the main trunk in the thyroid space, it may occur that only this posterior subdivision of the nerve is injured during surgical procedure. King-Gregg, in their article published in the *Annals of Otology, Rhinology and Laryngology*, December,

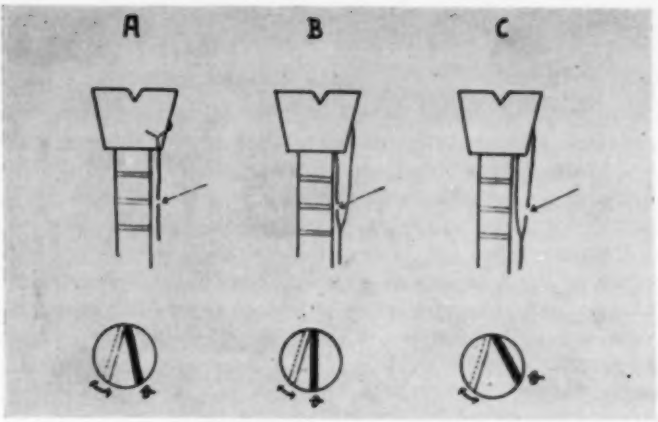
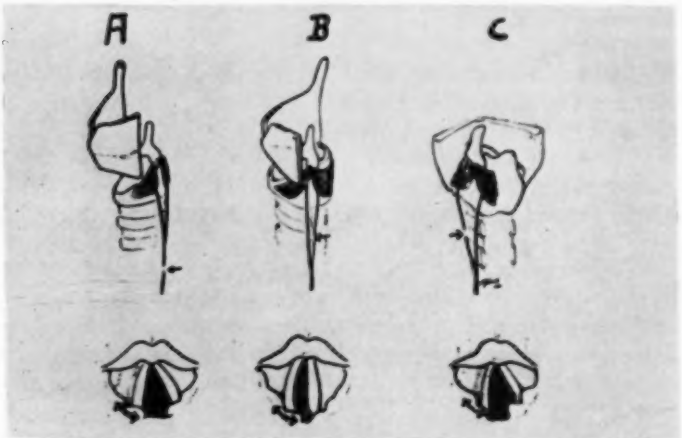


Fig. 2. Sketched schematically.



Explanation, according to King-Gregg, of the various positions assumed by paralyzed vocal cords after strumectomy, demonstrated on a case of injury to the left recurrent nerve at the level of the second tracheal cartilage.

Fig. 3. Sketched semi-schematically.

I. High Division—Lesion involving the entire recurrent nerve below the point of bifurcation.

Result: Total left-sided paralysis of the recurrent nerve. Laryngoscopically: Left vocal cord assuming an intermediate position with loss of tension (flaccid paralysis). This position is commonly called cadaveric position. Case A.

II. Low Division—1. Injury to the posterior ramus of the recurrent nerve (R. med.).

Result: Paralysis of the abductors, paresis of the posticus. Laryngoscopically: Left vocal cord fixed in midline position. Case B.

2. Injury to the anterior ramus (R. lat.).

Result: Paralysis of the adductors, lateral paresis. Laryngoscopically: Left vocal cord fixed in lateral position. Case C.

1948, describe how it is easily possible in thyroidectomies, providing the recurrent nerve divides in the thyroid space, to injure only one ramus without injuring the other. Thus, injury to the *anterior ramus* would affect the *adductors*. The same concept can be applied to injuries to the *posterior ramus* with the resulting paralysis of the *abductor*. Recurrent nerve injury to the main trunk of the nerve before it divides into anterior and posterior rami would paralyze the adductors, abductors and constrictors of the larynx. According to King-Gregg the majority of laryngeal paralyses is the result of nerve injuries during goiter operations.

In view of the above explanation we were faced with the following questions:

1. In normal subjects, what is the rate of cases presenting either "high" (endolaryngeal) or "low" (extralaryngeal) division of the recurrent nerve?

2. What is the rate of cases presenting an extralaryngeal motor branch (or ramus posterior) supplying the abductor?

It is easily understood that King-Gregg's conclusions can be evaluated only on the grounds of clear anatomical evidence, and only by means of minute studies and investigations conducted on a large scale does it seem possible either to accept or to reject the opinion of these authors.

Anatomical findings on the distribution of laryngeal nerves dates back to ancient times. This knowledge has been transmitted to us by Galen, Vesal, Rufus and others who have made successful attempts to clarify and explain this subject. It is easily conceivable that in the course of many Centuries their findings have been amplified as well as modified. Considering the limited space of this paper, it would lead too far to go into more details on this historical background; however, reviewing the recent literature on the distribution and function of the entire motor and sensory supply to the larynx, we have found in some instances a variety of terms which may cause confusion. Thus the term *high* or *low*

division, designating division of the recurrent nerve into the anterior and posterior rami does not indicate with complete accuracy the site of bifurcation. Instead of describing the branching as occurring *inside* or *outside* of the injury zone, we would recommend general use of the terms *extralaryngeal* or *endolaryngeal* division, *specifying by all means the number of the corresponding tracheal cartilage, in order to designate the exact level of bifurcation.* We consider this an important feature.

In the works and writings of European anatomists and laryngologists as well as in those of American authors, the above specification has been omitted. In the present paper we are, therefore, concerned with a closer localization of the site of branching of the recurrent nerve, as well as an understanding of its distribution, as we have worked it out; moreover, our investigations were conducted with the objective of defining the rate of either endo- or extralaryngeal division of the recurrent nerve in our material, since reports on this percentage have shown wide differences up to now.

During 1955 and early in 1956, the findings gathered on this subject from the literature were re-examined by us in a series of consecutive and complete studies conducted at the Anatomical Institute, Vienna, under the direction of Prof. Dr. Hayek, in collaboration with the First University Clinic for Ear-Nose-and-Throat Diseases, Vienna, headed by Prof. Dr. Schlander, in which we attempted to assert our own viewpoint on the problem of recurrent nerve paralyses. In order to arrive at a maximum of exactitude, all nerve preparations were carried out by one laboratory assistant, who worked with a binocular microscope. One hundred dissected nerves were prepared: 40 preparations were made *in situ*, and later 60 were done on isolated larynges. With reference to the extralaryngeal site of bifurcation the following statements are supported by the evidence of our material:

a. In 100 dissected nerves we found *extralaryngeal division in every instance* after microscopic preparation.

b. The division presented characteristic features; besides short branches supplying the trachea, esophagus and the

capsula of the thyroid gland, the main trunk of the recurrent nerve commonly gave off a weaker ascending extralaryngeal branch.

c. Concerning the level of ramification it was observed that the above mentioned extralaryngeal branch left the main trunk most frequently at the level of the first, sometimes at the second, and very rarely at the level of the third tracheal cartilage. Extralaryngeal division at the level of the clavicles, or the sixth tracheal cartilage, as described by Morrison and Rustad was at no time noted by us. In our material there was no division lower than the level of the third tracheal cartilage.

In the course of our investigations we soon realized that the crux of the problem lies in the anatomical identification of the ramus posterior. Occurring as the mesial branch of the inferior laryngeal nerves, it supplies the clinically important single abductor muscle of the larynx, *viz.*, the cricoarytenoideus posterior or posticus; hence it controls the vital function of breathing, which is clinically more important than the phylogenetically, much younger function of speech. Injury to the ramus posterior, caused either by surgical manipulation or any other trauma, results in paralysis of the abductors of the cords which will always be considered a serious symptom; in fact, it endangers life when occurring bilaterally. It is, therefore, of great importance both to the laryngologist and the surgeon to be familiar with the course of the recurrent nerve and its divisions.

In this connection the question arises, is the extralaryngeal branch of the recurrent nerve which pursues its course mesial to the cricoarytenoideus posterior and spreads on the surface of this muscle, the ramus posterior proper of the clinical literature? Has this branch indeed the function of a motor nerve attributed to it by American authors and others, a function which constitutes the basis and essential requisite of King-Gregg's explanation? Finally, is this branch really the muscle nerve supplying the posticus, or is it a mere mucous nerve forming a portion of Galen's anasto-

mosis? This anastomosis between the superior and inferior laryngeal nerves has long been known to anatomists.

At the beginning of our study, in conformity with the reported results of Piersol, King and Gregg, Morrison and Rustad, we believed the extralaryngeal branch of the recurrent nerve to be the ramus posterior proper, as designated by these authors; however, in the course of our investigations, we became doubtful about this identification. In fact, on the evidence of our material, the extralaryngeal branch was revealed to be, not the motor fiber innervating the posticus but a mere mucous nerve of the hypopharynx which could split and subdivide. While this was our experience in every instance of the 100 nerves dissected in this group of cadavers, we likewise observed in all of our cases, that this extralaryngeal branch was connected with the internal ramus of the superior laryngeal nerve. Thus it constituted the caudal portion of the *Ansa Galeni* or *Galen's loop.*

Conversely to the results of American authors, we were furthermore able to demonstrate that the muscle nerve proper, supplying the cricoarytenoideus or posticus, was given off the inferior laryngeal nerve *endolaryngeally*, at a point located about 1 cm. upward from its entrance into the larynx. In no case did this endolaryngeal branching occur any lower than at the level of the cricoid cartilage (exactly 9 to 11 mm. above the lower edge of the cricoid cartilage plate). The nerve passed between the cricoarytenoideus posterior and the perichondrium of the cricoid cartilage plate, ascending mesially and reaching the interarytenoid muscle above the upper edge of the cricoid cartilage. Thus, in 100 preparations of dissected recurrent nerves, we failed to detect a single case of an extralaryngeal division into two motor branches distributed one each to the abductor and the adductor muscles respectively. Besides branches distributed to the thyroid gland, trachea and esophagus, the recurrent nerve gave off extralaryngeally but one sensory branch to the *Ansa Galeni.* After its entrance into the larynx, the inferior laryngeal nerve divided into a mesial and a lateral branch, the mesial spreading to the posticus and the inter-

arytenoid muscle, the lateral branch supplying the other internal laryngeal muscles (see Figs. 4 and 7). According to the above described anatomical evidence this appears to be the general functional pattern, exceptions excluded.

Our findings on this subject coincide with those of Williams, 1951, who reported extralaryngeal division of the recurrent nerve into motor and sensory fibers, but did not admit the occurrence of an extralaryngeal division into abductor and adductor branches. We are also in agreement with the opinion expressed by Lanz and Wachsmuth in their "Textbook of Practical Anatomy," 1955. These authors state that division of the recurrent nerve into a ramus dorsalis and ventralis respectively, occurs only after entrance of the main trunk of the nerve into the larynx. The French author, Lazorthes, in 1955, described ramification of the recurrent nerve in the same manner.

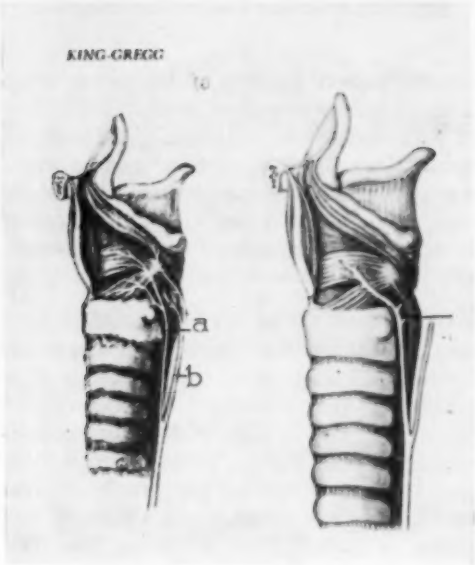
ANATOMICAL FINDINGS.

A. The extralaryngeal division of the recurrent nerve in our material could be identified as a division in motor and sensory fibers; it occurred at the level of the first, second or third tracheal cartilage.

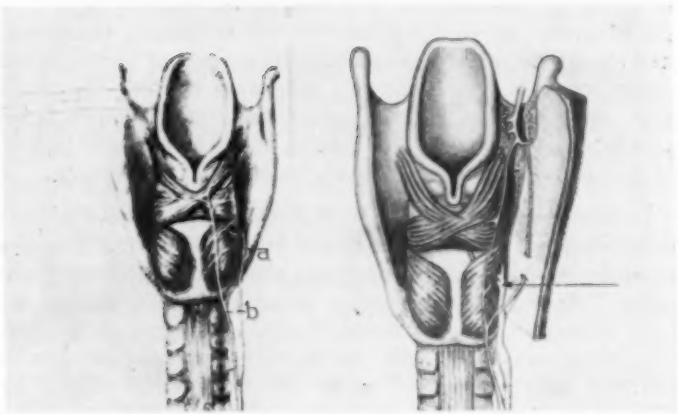
B. At the point of bifurcation the recurrent nerve left off a sensory branch, which was distributed to the mucous membrane of the hypopharynx, then split and subdivided, forming an anastomosis between the inferior and superior laryngeal nerves. It thus constituted the caudal portion of Galen's loop. This nerve was located *on* the posticus.

C. In every instance, the ramus posterior proper branched off only endolaryngeally (see Figs. 4 and 7). The division of the inferior laryngeal nerve into the posterior ramus (the mesial branch), and the anterior ramus (the lateral branch) occurred in all cases about one cm. above the lower edge of the cricoid cartilage. The ramus posterior proper (See arrow!) was located *below* the posticus (between the muscle and the perichondrium).

From the above statements it is evident we could not trace



Figs. 4 and 5.



Figs. 6 and 7.

At the left: Figs. 4 and 6, reproduced from King-Gregg's paper, showing extralaryngeal division (below the larynx) of the recurrent nerve into two main branches, one of which is distributed to the abductor muscle, the other to the adductors.

At the right: For comparison, Figs. 5 and 7, corresponding to Figs. 4 and 6, sketched schematically, in the same size and proportion. They illustrate the conditions encountered in our study, viz., our anatomical findings based on 100 microscopical preparations of recurrent nerves.

any low extralaryngeal division of the recurrent nerve into abductor and adductor branches respectively; however, the presence of this anatomical condition constitutes an essential requirement of King-Gregg's solution; hence, only if *Galen's loop* really carries motor fibers, a certain clinical significance must further be attributed to King's explanation. Most authors, though, on the grounds of their physiological results, express serious doubts concerning the motor innervation of *Galen's loop.*

Conflicting views likewise have been expressed by the previously mentioned authors on the frequency of occurrence of a low extralaryngeal division. Reports vary from 11.6 to 78 per cent. In view of such wide differences of opinion, general statistics should also be given due consideration. In compiled statistical findings on paralyzed recurrent nerves after strumectomy, there appears more than twice the number of midline or paramidline positions than intermediate positions of the cords (Kecht); moreover, the experience gathered in the course of practical endoscopy is more likely in favor of the opinion that the extralaryngeal branch of the recurrent nerve distributed on the surface of the muscle and closely below its mucous membrane, cannot be the motor nerve supplying the posticus; otherwise it would be much more sensitive, for instance, to pointed foreign bodies aspirated into the esophagus and lodging at the level of the larynx, or to rough handling during endoscopy in this area.

In summary, we point out that we must caution against overrating the clinical importance attributed to the presence of an extralaryngeal motor ramus posterior to the recurrent nerve. Fully sustained by our anatomical observations, we have serious reasons to doubt that the problem of the baffling behaviors of the vocal cords after thyroidectomy has as yet been satisfactorily solved by the explanation offered by King and Gregg, hence we believe that any definite statement to this effect would be premature.

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SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The date for the acceptance of papers and films for the program of the Sixth International Congress of Otolaryngology has passed, and the scientific program has been completed. 238 papers will be presented simultaneously in four sections during each General Session. These represent contributions from otolaryngologists in 35 countries and deal with a remarkably wide variety of subjects in the specialty.

Applications for the presentation of films have been sufficient to provide 18 hours of motion pictures to run simultaneously with the presentation of scientific papers.

Forms for requesting hotel registrations in Washington have been distributed to all who have registered as members of the Congress. Those wishing to register should communicate with the General Secretary, 700 N. Michigan Avenue, Chicago 11, Illinois, U. S. A. Applications for hotel accommodations will be sent as soon as registration is completed.

PATHOLOGIC CHANGES IN THE INNER EARS OF SENILE GUINEA PIGS.*†

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J. B. ROGERS, M. D.,
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Pathologic changes that occur with old age in animals have in many instances aided in a better understanding of the nature of the process in humans. Those described for the inner ears of cats[1] and dogs[2] have been found to be similar to the degenerative lesions in the cochlea of the aging human.[1,3,4,5] Two different types of atrophic changes have been recognized: *1.* an atrophy of the neural elements of the cochlea; *2.* an atrophy of the epithelial and neuroepithelial cells of the scala media. The two may occur simultaneously in the same specimen. They have been found to be independent of vascular pathology although in some instances the latter may be a contributing factor. The basal coil of the cochlea is affected first, and this location of the lesion is correlated with the high-tone hearing loss of presbycusis[6].

Some laboratory animals are not suitable for a study of senile changes in the inner ear, viz., the white rat, for which the incidence of chronic middle ear infection is high, even in well established colonies. The guinea pigs used in this study were supplied from the colony of Dr. James B. Rogers of the University of Louisville for which lineage, breeding data, weight records and so forth were available. None of the specimens revealed middle ear infection or labyrinthitis

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as did guinea pigs of two or more years in age that were purchased on the open market. The senile animals appeared old. The nails were long and curved, the hair less dense and in most instances the guinea pig was less alert. They were selected from the colony on the basis of a diminished or absent Preyer's reflex.

Another reason for using the guinea pig for this study was the amount of information that is now available relative to the physiology and pathology of the guinea pig's cochlea. Electrophysiological measurements[7] on the same animals and a control group of young animals from the same colony were made at Central Institute for the Deaf, St. Louis, Mo. The results of these studies will be reported separately.

MATERIAL AND METHODS.

Thirteen guinea pigs ranging in age from one year and 356 days to four years and 209 days comprised the group of older animals for this study. There were three males and ten females. Six young guinea pigs of the same stock (Rogers) served as a part of the controls and ranged in age from 39 to 93 days. Three of these were males and three females. Ten guinea pigs of local stock (Haskins) ranging in age from four to eight weeks served as the remainder of the controls. The right ear of each of these animals was opened and electrophysiological measurements recorded. The animals were then perfused with physiological saline solution to wash out the blood. This was followed by Heidenhain Susa solution for fixation. The temporal bones were then removed and the specimens decalcified, dehydrated and embedded in celloidon according to the procedure used for other studies (Covell[8]). Serial sections of 15 microns in thickness were stained with Harris' hematoxylin and eosin and mounted in Canada balsam.

Mid-modiolar sections of the cochlea served as a guide by which the extent of changes on either side of the modiolus could be observed. These changes were charted according to the method used for the study of the effects of intense

sound in the cochlea[8]. In order to determine the relative number of spiral ganglion cells in each turn of the cochlea counts were made on sections that passed through the axis of the modiolus. It was necessary to use most of the slides in the series for this purpose. An average of counts on four to six consecutive sections through each turn usually sufficed for a comparative count. Only those cells were counted for which the section passed through the center of the cell. The latter was determined by focusing with the fine adjustment of the microscope within .005 mm in either direction, as read on the scale of the hand screw. A hand counter was used to count the cells. The magnification used was approximately 300 times. The counts on four specimens were made by two different individuals. An average value of approximately five per cent less in total number of cells was obtained by one person as compared to the results of a more experienced microscopist.

FINDINGS.

The Modiolus. Within the modiolus blood vessels with their perivascular spaces and tissue lie between bone and nerve. They are arranged in a spiral fashion from base to apex of the cochlea. No discernible changes were observed in the walls of any of the vessels of older animals although there was an increase in the amount of perivascular tissue over that usually seen in young animals. The nature of this tissue is loose to fairly compact connective tissue with what appears by ordinary staining methods to be cells of the pia and probably arachnoid. Small tufts of these cells in some instances, project into the perivascular spaces. Pigment cells within the walls of the blood vessels are not unusual. Areas in which there is evidence of increase in perivascular elements are frequently associated with an osteitis of the bony wall of the modiolus. While this is not a feature dependent entirely upon age of the animal it is found to be considerably increased with age. The animals of two to three years had more of this reaction than younger animals, and those of four to five years of age had considerably more new bone formation. It was often found in larger amounts

in the lower to middle part of the first turn and at the level of the third and fourth turns. The modiolus at the levels of the second and lower part of the third turns showed less of this reaction and when present new bone was located on the apical surface of the osseous margin of Rosenthal's canal.

Spiral Ganglion Cells and Peripheral Nerve Fibers. The predominant pathologic change in sections through the coch-

TABLE I.

PERCENTAGE VARIATIONS OF SPIRAL GANGLION CELLS FOR AGED GUINEA PIGS AS ESTIMATED FROM THE MEAN VALUES DETERMINED FOR YOUNG GUINEA PIGS.

Animal Number and Sex	Age (days)	Turns of the Cochlea* 1 rw-m	1 u	2 l	2 u	3 l	3 u	4 l-u
928 F	721	-28	13	12	—	—	-35	-41
931 F	770	-65	11	—	9	8	—	-77
924 F	805	-16	—	-10	-25	-37	-31	-46
927 F	811	-28	-16	—	—	-9	—	-62
926 M	848	-50	-26	-36	-11	-29	-26	-30
925 F	1023	-41	-15	—	-5	-13	-9	-45
948 F	1541	-58	-34	-5	-21	-40	-28	-72
947 M	1576	-54	-7	-16	-21	-60	-46	-61
949 F	1669	-59	—	-41	-51	-73	-88	-94

*The turns of the cochlea are 1, 2, 3 and 4 with "l" for the lower portion and "u" for the upper part. In the lower part of the first turn "rw" is the area nearest the round window while "m" is the middle part of the first turn. Separate estimates could not be made of the number of spiral ganglion cells in the most basal portion of the first turn and in the fourth turn because the ganglion in these areas is fused into a group of cells.

leas of senile guinea pigs was the fewer number of ganglion cells. This loss was most apparent in the apex and less near the base of the first turn. The intervening turns showed a relatively normal number of ganglion cells, except for the three oldest guinea pigs and one of 848 days. The results of counts of number of ganglion cells for each turn are shown as percentage values in Table I. These estimates are based on counts made for sections of cochleas of young guinea pigs for which the percentage value was taken as 100. The loss in the apex of the cochlea for nine specimens shows a range of 30 to 94 per cent for the fourth turn, while the base of the cochlea (lower and middle portions of the

first turn) shows a range of 16 to 65 per cent. For five senile animals a count was made of cells for right and left cochleas, and in only one instance was there a significant difference between the counts for the two ears. It is evident from the range in ages of the animals that an increase in age is associated with further degenerative changes in ganglion cells. Figs. 1 and 2 represent mid-modiolar sections through cochleas of young and senile guinea pigs, respectively. In the latter there is a loss of ganglion cells chiefly in the fourth turn and with less marked changes in the lower part of the first turn. There is apparently nothing distinctive in the manner in which the spiral ganglion cells undergo degenerative changes with senility. The process consists usually of an eccentrically placed nucleus, chromatolysis and loss of ground substance. Later there is diminished stainability of nucleus and nuclear substance usually with some degree of swelling, and loss of cytoplasm, cell membrane and nucleus. Accompanying the disappearance of spiral ganglion cells is the presence of a few neuronophages. These are usually not more than two to six in number. Similar cells appear at the periphery of ganglion cells in control material and resemble in size and location the so-called "satellites" of cortical neurons of the central nervous system. They are undoubtedly the cells responsible for the final removal of the degenerated ganglion cell and are probably a glial-type of cell. A comparison of the control material with that from senile guinea pigs gives the impression that there is an increased satellitosis in the latter. Whether it is absolute or relative to the loss of spiral ganglion cells is a problem for further study. In Fig. 3 is shown the loss of ganglion cells and nerve fibers in the third and fourth turns of the cochlea of a guinea pig, 1669 days of age. The lower basal turn of the same cochlea (see Fig. 4) shows considerably more ganglion cells.

The loss of peripheral nerve fibers accompanies the degenerative processes associated with the spiral ganglion cells. Degeneration of the proximal and distal fibers of the bipolar cells proceeds in each direction, and the last myelin

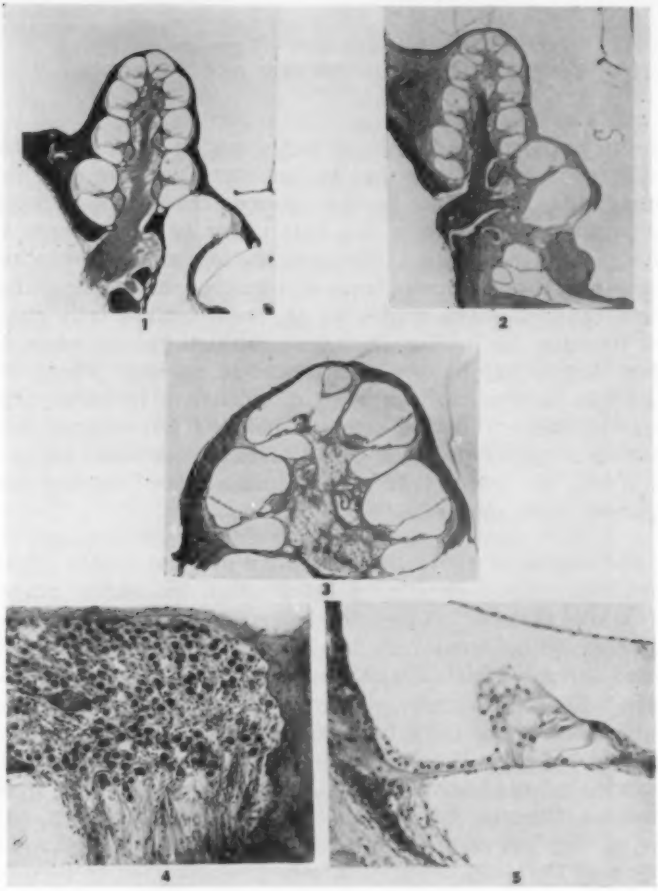

Fig. 1. Photomicrograph of a mid-modiolar section through the right cochlea of a control guinea pig, 4½ weeks of age, showing the distribution of spiral ganglion cells. Heidenhain Susa fixation, Harris hematoxylin and eosin stain. X-14.5.

Fig. 2. Photomicrograph of a mid-modiolar section through the right cochlea of a guinea pig, 811 days in age. There are fewer ganglion cells in Rosenthal's canal in the fourth turn and a less marked loss in the lower part of the first turn. X-14.5.

Fig. 3. Photomicrograph of a mid-modiolar section through the two upper turns of the right cochlea of a guinea pig, 1669 days in age. Only a few spiral ganglion cells remain. There is also a marked degenerative change in the peripheral nerve fibers. The organ of Corti is flattened with degeneration and loss of sensory cells and collapse of supporting structures. In the fourth turn the tectorial membrane appears as a homogeneous eosinophilic mass. The stria vascularis is markedly atrophic. X-45.

Fig. 4. Photomicrograph of a section through the lower basal turn of the same cochlea as shown in Fig. 3. Many of the ganglion cells are lost while others are undergoing degenerative changes. There was an estimated loss of approximately 59 per cent for this region as compared to 94 per cent for the fourth turn. X-250.

Fig. 5. Photomicrograph of a mid-modiolar section through the upper part of the third turn of the scala media of the same cochlea as Fig. 2. There are degenerative changes in the hair cells. Some of the external hair cells are lacking while the internal appears deeply stained and somewhat shrunken. There are noticeably fewer mesothelial cells. X-250.

sheath to disappear distally is in the region of the habenula perforata or centrally in the substance of the cochlear nerve; thus it does not seem to precede the loss of ganglion cells but instead follows it. This is somewhat contrary to the present concept of the sequence of events for other factors, such as acoustic trauma, resulting in similar pathologic changes. The nuclei of the neurolemma cells show no tendency to increase in numbers; but instead some of them lose their ability to stain, and in an area where the ganglion is practically depleted of cells they frequently appear as "ghosts". The efferent bundle (Rasmussen) was lacking a few fibers in the basal turns of the oldest animals and was not present in apical turns where degenerative changes were marked.

The Organ of Corti. Degenerative changes in the organ of Corti is not a predominant feature in the earlier stages of senile changes. They do, however, become more pronounced with increase in age of the animal and only for those turns associated with marked loss of spiral ganglion cells. Fig. 5 illustrates the type of changes found in the upper part of the third turn of the right cochlea of an 811-day old animal. The two most external hair cells are in an advanced degenerative state, Deiters' cells show less pronounced changes, the internal hair cell is shrunken and stains deeply and the mesothelial cells directly beneath the organ of Corti are fewer in number. In the fourth turn of the same specimen the degenerative changes for the organ of Corti were more marked, particularly in the supporting cells. The basal turn of the cochlea did not show the same amount of degenerative lesions, in fact, the upper and middle portions of the first turn were within normal limits. Another histological method might reveal changes which remain obscure by the present method. The degenerative changes of the three oldest animals consisted of a loss of supporting and sensory cells with a collapse of the tunnel rods (upper part of the second turn and including the fourth turn). The organ of Corti in the round window region was similarily affected, but in the remainder of the first and second turns the organ of Corti showed remark-

ably few changes. The loss of mesothelial cells is considerably more widespread than changes in the organ of Corti for all senile animals. An incomplete covering of the basilar membrane on the surface of the scala tympani was found to occur for all turns except in the first turn even in the presence of a relatively normal appearing organ of Corti. The mesothelial cells are usually more numerous in this region for control specimens than in the turns above.

Stria Vascularis. The stria vascularis was without pathologic changes for many of the specimens. When present they consisted of atrophic changes, increased pigmentation, and cystic degenerative changes. No consistent finding was present for all senile animals, and in one of the older animals the stria vascularis was atrophic in the second to the fourth turns inclusive, with increased pigmentation and cystic degeneration in the first turn. In this particular specimen other findings, such as for the organ of Corti, were in a similar atrophic state. One of the control animals showed cystic changes in the middle of the first turn, and another revealed a localized area of atrophic stria in the lower part of the second turn. Pigmentation is likewise a variable factor in control animals. Degenerative changes in the stria vascularis and increased pigmentation while they may not always accompany the aging process in the guinea pig do occur more frequently in senile animals and become more pronounced with increase in age.

DISCUSSION.

In view of the lack of suitable material from other strains or colonies of senile guinea pigs it is advisable to restrict the pathological findings as described to the present series of animals. There were, however, no discernible differences in younger or control animals of the same colony and those procured from other sources. It is doubtful that the pathological changes are peculiar to this colony (Rogers) of guinea pigs.

The results of the present studies are in agreement with the findings of other investigators as to the general type

of pathologic changes in the inner ears of senile animals and humans. The chief difference is the location of the pathology in relation to the turns of the cochlea. The guinea pig has four turns and the cochleas of the other animals for which changes have been reported have approximately two and one-quarter turns. The reason for the occurrence of these changes apically, with usually less in the lower part of the basal turn, is not known. One might expect the vascular supply to vary sufficiently in different species, particularly with four turns to the guinea pig's cochlea, to explain this difference. There is, however, no vascular pathology that is consistent with the occurrence of these lesions.

Further studies may reveal that modiolar changes are not independent of those in ganglion cells and possibly the organ of Corti. The frequent finding in the cochleas of the older animals of rather extensive osteitis of the bony wall of the modiolus, cystic perivascular areas and occasional increase in amounts of perivascular tissues in other regions indicates some disruption of normal function. Osteitis in the fundus of the internal auditory meatus of the human temporal bone has been described[5] at the point of exit of the cochlear nerve fibers. There are several differences in modiolar structure between the cochlea of the guinea pig and that of the human. The cochlear nerve in the guinea pig leaves the modiolus through a single opening, but bony partitions with foramina are located proximal to the spiral ganglion. Such an arrangement may be a contributing factor to the location of degenerative changes in the apical turns of the cochlea.

It is apparent that the two types of atrophy, neural and epithelial, are independent of each other at least in the earlier stages. They may be present in the same turn of the cochlea, but loss of ganglion cells, except when extreme, is usually not accompanied by marked degenerative changes in the organ of Corti.

The recent cytochemical studies of Hammer[9] on spiral ganglion cells of the guinea pig's cochlea, subjected to

acoustic trauma, showed a decrease in protein fraction and cytoplasmic RNA fraction with no changes in the lipid fraction immediately following exposure. Two months later there was reduction in number of cells and cell volume, a further decrease in protein content and a higher concentration of RNA about the nucleus. Six months after the exposure there was further reduction in cell numbers, and the protein content was approximately one-third of the normal value. These degenerative changes are considered to be secondary to an interruption of the innervation of the end organ. The degenerative changes in the ganglion cells are similar to those that occur with senility; however, interruption of the innervation cannot be considered as the predisposing factor in senility. In the latter the ganglion cell seems to be primarily affected and the peripheral nerve fibers secondarily. The efferent bundle (Rasmussen) is also subject to degenerative changes as described by Schuknecht[1] for senile cats and somewhat less in the present series of guinea pigs' cochleas. The efferent bundle has been shown by Fernandez[10] to be spared of degenerative changes in experimental acoustic trauma.

The removal of the degenerating spiral ganglion cells by neuronophagia has been described by Saxen[4]. The process as observed for the guinea pig is similar but at no time are there more than a few cells undergoing active neuronophagia in an area of degenerating cells. The neuronophagocytes are probably glial in type. They are undoubtedly present in the ganglion as "satellites" and become phagocytic with degeneration of the nerve cell.

The embryonic nature of the spiral ganglion cell has been emphasized by Hamberger and Hydén[11]. There are several peculiarities about the inner ear that reflect an embryonic type of structure such as the enchondral layer of the otic capsule that is regarded as representing a stage in arrested development of bone in cartilage. If the immaturity of the ganglion cell is a factor in susceptibility to degenerative changes of old age then it might be expected that the whole ganglion would be involved simultaneously

instead of particular regions such as the base of the human cochlea. At present there is no satisfactory evidence to show that cells of the spiral ganglion vary in their appearance and histochemical composition with the different turns of the cochlea.

SUMMARY.

The histologic changes observed in the stained sections of the cochleas of senile guinea pigs as compared to those of young animals from the same colony and also from other sources were: *1.* A diminished number of spiral ganglion cells in the third and fourth turns of the cochlea with subsequent degeneration of peripheral nerve fibers. There were similar but less marked changes in the lower part of the first turn. The intervening turns except for the three oldest animals and one of 848 days, were without a significant loss of spiral ganglion cells. *2.* Neural atrophy preceded atrophic changes in the organ of Corti. The latter was severe only in the oldest guinea pigs of four years and more in age. Loss of mesothelial cells was a common finding that increased with age of the animal. *3.* Osteitis and changes in perivascular tissues of the modiolus become more pronounced with age. *4.* The principal difference between the lesions found for the guinea pig and those previously described for the human and other animals is the location of the more marked changes in the apical areas of the guinea pig's cochlea instead of the lower part of the basal turn. *5.* The results are to be regarded as specific only for this particular strain of guinea pigs.

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NEW ELECTRO-LUNG.

An electrical device for stimulating and regulating the muscles of breathing is giving cerebral palsied children a chance for more normal speech. The apparatus, known as an electro-lung, is being used on children whose breathing rate is disturbed so much that they have difficulty in speaking.

The device is in use in the Northwestern University department of speech correction and audiology. According to Dr. Harold Westlake, head of the department, normal persons breathe approximately 14 to 16 times a minute while some children with cerebral palsy breathe between 60 and 80 times a minute. Such rapid breathing makes continuous speech impossible, he says.

Work on cerebral palsied children at the Northwestern speech clinic has produced excellent results within six weeks, according to Dr. Frank Wilson, research worker. He reports that six weeks of treatment for one-half hour a day changed one child from being totally inarticulate to having "socially acceptable" speech. In most cases the improvement is moderately good. Dr. Wilson refers to the electro-lung as a "movable respiratory brace."

DETECTION OF MALINGERING BY PERIODICALLY SWITCHED SPEECH.*

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The logic premises for this new test for the detection of simulated deafness derive from the results of studies on the intelligibility of periodically interrupted speech and of continuous speech switched periodically from one ear to the other.

Cherry and Taylor[1,4] have observed that when a recorded message (at a rate of about 85 words per minute) is applied to one or the other ear of a subject and switched periodically, so as to keep a 50 per cent on-off ratio, the intelligibility of the said message is nearly perfect for all switching frequencies (see Fig. 1). A lower articulation score, which, however, varies in different subjects, has been noticed by the same AA. only when the message is read at a higher syllabic rate (138 words per minute), and only for a switching rate of 0.2-0.3, keeping the 50 per cent on-off ratio unaltered. This could be considered as a good objective measure of the reaction time needed to transfer the attention from one ear to the other. These data have been confirmed by our personal researches; in addition we have noticed an increased subjective difficulty even for the recognition of a message recorded at a speed of 85 words per minute for a switching period of 0.2-0.3, while at all other switching rates it is understood correctly; furthermore, we have observed that the threshold shift for continuous speech, when it is switched periodically from one ear to the other, is very small (5-10 db), and we have been able to ascertain that the listener cannot refer to one or the other ear the sensation of the various fractions of the switched speech, the sensation being that of interrupted speech heard binaurally.

On the other hand, the researches performed on interrupted speech (with a 50 per cent on-off ratio) have shown that its

* From Ear, Nose and Throat Clinic of the University of Milan (Italy). Director: Prof L. Pietrantoni.

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intelligibility varies considerably, depending on the frequency of interruption. The results reported by Bocca and Camisasca[2] and by Miller and Licklider[3] do not substantially differ. The curves reported on Figs. 2 and 3 show how the intelligibility of speech, which is absent or poor for low interruption rates, quickly rises with the increase of the rate of interruption, and approaches 100 per cent for an interruption rate of 8-9 per second. It appears logical that the minimum of intelligibility corresponds to one interruption per second, as the "on" period is roughly equal to the duration of one word spoken at a normal rate of speech, while the maximum of intelligibility at an

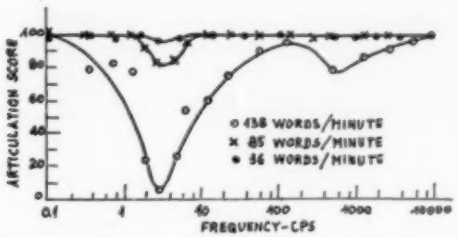


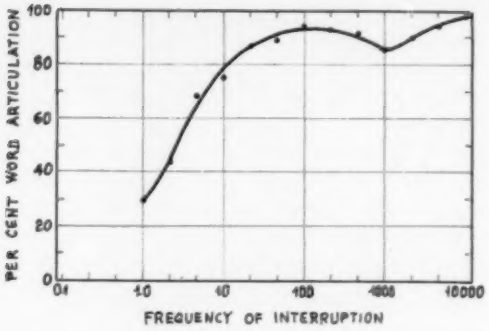
Fig. 1. Articulation scores for continuous speech, switched periodically from one ear to the other. For each ear the proportion of the period occupied by speech is 50 per cent, the remainder being silent. Results for three rates of speaking are given.

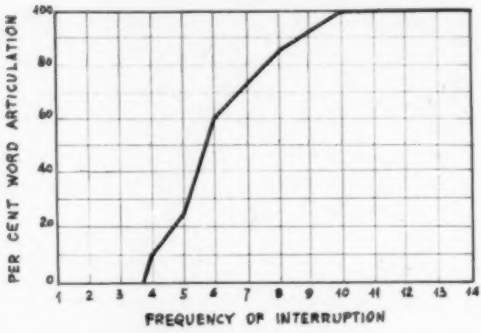
interruption rate of 10 per second may be also attributed to the temporal characteristics of the words.

We have taken advantage of the different effects on speech intelligibility of a periodical switching or interruption, for the detection of simulated unilateral deafness.

DESCRIPTION OF THE TEST.

We have used a common audiometer (Mod. Elit 811-B) coupled to Bocca's periodical interrupter. The latter, which is schematically illustrated in Fig. 4, consists of a mechanical relay commanded by an electronic oscillator. The passage from interruption to switching is obtained through a simple commutator. The switching or interruption rate (always with a 50 per cent on-off or on-on ratio) can be varied from a min-





Figs. 2 and 3. Word articulation as a function of rate of interruption for a speech-time fraction of 0.5. (Fig. 2: after Bocca and Camisasca; Fig. 3: after Miller and Licklider).

imum of two to a maximum of twelve per second. A higher switching rate, which can be obtained only by means of electronic devices, is not relevant to our purpose.

The speech material used in this test was composed of several lists of meaningful short sentences, recorded at an average speed of 85 words per minute, and was presented to the subject through headphones.

After having determined the hearing threshold in both ears for pure tones, we immediately use the switched speech test, after inviting the subject to repeat the message he hears "in a peculiar way" in the ear stated to be healthy; thus making him

believe that the examiner wants to test the hearing function of that ear. This is possible due to the particular subjective sensation of switched speech, as reported above. The intensity of the alternated message is established at an intensity level about 30 db higher than the hearing threshold in the healthy ear.

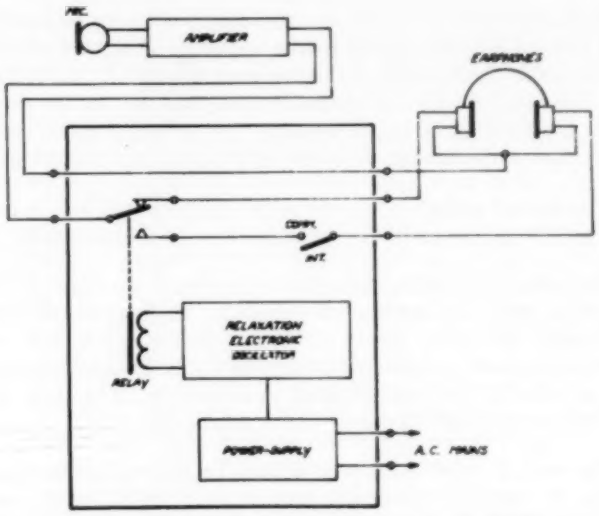


Fig. 4. Periodic switch (commutator or interrupter).

If the listener is actually deaf on one side, the message will reach the healthy ear as interrupted speech, and the articulation curve will rise from 0 per cent to 100 per cent for an increase from two to eight per second of the switching rate. If, at a switching rate of 8 per second, the subject states that he is not able to understand the message, he is clearly simulating; but we cannot get any information as to the state of hearing in the allegedly deaf ear.

If, on the contrary, 100 per cent discrimination is obtained at a siwtching rate of 2-3 per second, at an intensity level 30 db higher than the hearing threshold in the healthy ear, it is

beyond doubt that the hearing loss in the allegedly deaf ear is not greater than 20 db, *i. e.*, this ear is practically normal. As a matter of fact, the malingerer has no cues as to the provenance of the switched speech and states candidly that he is able to understand at a low switching rate, being unaware that the "deaf" ear is actually participating in the overall discrimination.

It may happen that 100 per cent discrimination at a switching rate of 2-3 per second is attained only at a level of intensity higher than 30 db above normal threshold: in this case the hearing loss in the allegedly deaf ear will be 10 db less than the reading of the attenuator, 10 db being the threshold shift for periodically switched speech, as mentioned above.

The test of switched speech can thus allow not only a qualitative but also a quantitative estimation of malingering.

The pure tone audiograms shown in Fig. 5 are those of three subjects, who, on occasion of a military medical examination, pretended to suffer from unilateral deafness. A test with switched speech, performed according to the method described, led at once to the diagnosis of malingering. The data were confirmed later by other classical malingering tests.

The test of switched speech is based upon well-defined principles of acoustic physiology, and in every case and for every subject it offers unmistakable and clear-cut results; furthermore, by this test, it is possible to eliminate an important factor of uncertainty due to the particular psychic conditions of the examined subject. As a matter of fact, the malingerer, who knows that the advantage he seeks could be neutralized by the discovery of a simulation, approaches the hearing test in an apprehensive state, thus rendering the known tests very delicate to perform and their results very difficult of appreciation.

On the contrary, with the switched speech test, complete collaboration of the patient is easily gained, as he believes that the examiner is going to test the hearing function in the healthy ear.

Finally this test allows a quick diagnosis by means of a

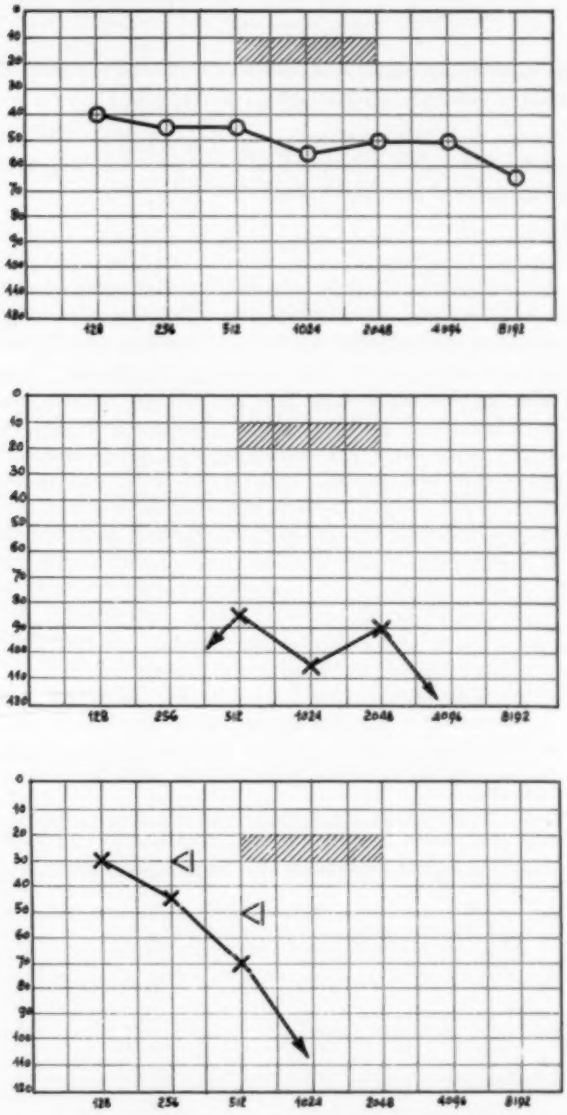


Fig. 5. Hearing thresholds for tones by air conduction and hearing thresholds for switched voice in three malingerers. The shaded area represents the level of intensity at which 100 per cent of switched sentences were correctly repeated.

simple and cheap apparatus, which may become a part of every common audiologic equipment.

Due to the above reasons, we believe that the switched speech test should be adopted as a current method of diagnosis of unilateral simulated deafness.

SUMMARY.

Here is described a new test for the diagnosis of simulated unilateral deafness, based upon the difference of intelligibility of a message periodically switched from one ear to the other or periodically interrupted in one ear. If one ear is actually deaf, the normal ear listens to a periodically interrupted message, and the discrimination will gradually rise from 0 per cent to 100 per cent when the switching rate increases from two to eight to nine per second. If, at a switching rate below three per second, the discrimination approaches 100 per cent, this clearly indicates that the alleged deaf ear can perceive the fraction of the message which does not reach the opposite ear; that is, the ear is not deaf. The intensity level at which 100 per cent discrimination is obtained by a switching rate below three per second will roughly indicate the amount of hearing loss in the ear where deafness is simulated.

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BRONCHOGENIC CARCINOMA: DIAGNOSTIC ASPECTS OF 228 PROVED CASES.*†

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In recent years emphasis has been placed on the early diagnosis of pulmonary disease due to the rising incidence of lung cancer and the severe nature of this lesion. Mass chest surveys stress the increasing frequency of occurrence of bronchogenic carcinoma in white males over 45 years of age; yet there has been a steady decline in the death rates from other pulmonary diseases in men of this age group. The incidence of lung cancer is showing a more rapid increase than that of any other malignant neoplasm.[1]

Upon the bronchoscopist has fallen the responsibility of proving or disproving the existence of lung neoplasm prior to thoracic surgery. While bronchoscopic biopsy formerly would show positive results in 60 to 70 per cent of cases according to published reports[2,6] the rate is now much lower. Unfortunately, in many patients with lung cancer, the direct bronchoscopic examination shows little evidence of neoplastic disease; therefore, it has become increasingly important to utilize all the methods of bronchologic diagnosis at our disposal, in order to secure early definitive treatment for the lung cancer suspect.

The material for this study was derived from an analysis of 228 cases of bronchogenic carcinoma at operation or autopsy in which bronchoscopic examination was performed.

INCIDENCE.

Bronchogenic carcinoma is encountered primarily in patients in the fifth, sixth and seventh decades of life. In our

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series, 91 per cent of the patients were between 40 and 69 years of age. The oldest was 80 and the youngest 31 years of age. There were 200 men (87.7 per cent) and 28 women (12.3 per cent). All of the patients were of Caucasian descent.

ETIOLOGY.

Bronchogenic carcinoma remains a disease of obscure etiology although many studies of its cause are going forward. Controversy still rages over the relationship of cigaret smoking to the cause of this disease. As pointed out by Hueper,[7] the total epidemiologic, clinical, pathologic, and experimental evidence at hand clearly indicates that not a single, but several if not numerous, atmospheric pollutants are responsible for the cause of lung cancer. The additive, cumulative and synergistic effects of several recognized respiratory carcinogens in the production of cancers in the general population are a distinct possibility. On the other hand, restricted industrial groups may be exposed to well defined and highly potent respiratory carcinogens. It is believed that the recent alarming rise in lung cancer in males is causally related to the local and general development of modern industry and the use of its products. It may be possible that cigaret smoking has played a contributory role, but the total evidence available does not favor the concept that cigaret smoking represents a major factor.

On the other hand, Hammond[8] states that cigaret smoking increases the probability that lung cancer will develop in the individual but that smoking is not the only cause of the disease, although it is an important factor.

In the present series of 228 proved cases of bronchogenic carcinoma, 89.9 per cent of the patients smoked; 150 patients (65.7 per cent) smoked 20 or more cigarets daily, or an equivalent amount of pipe or cigar tobacco for periods ranging from 20 to 45 years. Eighty-nine per cent of the patients in the group were city dwellers and lived in an environment in which the atmosphere contained varying amounts of possible carcinogenic dusts, gases, fumes and chemicals. Sixty-

five per cent of the patients had worked predominantly indoors, while 35 per cent had outdoor occupations.

PATHOLOGY.

From the clinical point of view, pulmonary cancers may be divided into two groups: those found near the hilus involving the major bronchi, and those which appear in the periphery of the lung. Epidermoid and undifferentiated carcinomas tend to originate in the lobar or segmental branch bronchi and are centrally located. Adenocarcinoma, on the other hand, usually originates in a more peripheral location.

In the present series of 228 patients, there were 103 instances of squamous cell carcinoma, 77 cases of undifferentiated carcinoma, and 48 patients with adenocarcinoma. The squamous cell and undifferentiated carcinomas constituted over three-fourths of the group. Only a small percentage of the squamous and undifferentiated carcinomas were found in women. In adenocarcinoma, a more nearly equal distribution was found.

The right lung was involved slightly more often than the left. One half the patients had lesions in the upper lobes, a factor of importance to the bronchoscopist.

SYMPTOMS AND SIGNS.

There are no symptoms pathognomonic of cancer of the lung. The onset is insidious and, as pointed out by Ochsner[9], Overholt and Schmidt[10] and Paulson and Shaw,[11] there is a "silent phase" of the disease when there are no symptoms until the bronchial mucosa is irritated or ruptured.

The commonest initial symptoms in this series were cough, which occurred in 82 patients (36 per cent), thoracic discomfort or "sense of heaviness in the chest" in 38 patients (17 per cent), and persistent symptoms following a chest cold, influenza or pneumonia in 49 patients (21 per cent). Shortness of breath, hemoptysis, wheezing and systemic symptoms occurred less frequently. In three cases the can-

cer alarm came from the discovery of an abnormal shadow on the routine chest roentgenogram.

In many instances, the patient had consulted his family physician, who usually prescribed antibiotics until more grave symptoms appeared. All too often, definitive diagnostic measures were delayed for an inexcusable period. The medical profession in general does not frequently enough consider bronchogenic carcinoma a possibility in men over 40 years with a lingering cough, vague chest discomfort or whose roentgenograms show "unresolved pneumonia", "atypical pneumonia", "viral pneumonia" or "non-specific pneumonitis".

As has been previously reported,[12] early bronchogenic carcinoma produces few physical signs. Later, signs of partial or complete endobronchial obstruction, ulceration, infiltration and secondary infection occur; therefore, obstructive or compensatory emphysema, atelectasis, asthmatoid wheezing, lobar or unresolved pneumonia, localized pneumonitis or effusion, are among the thoracic findings warranting a more complete investigation.

DIAGNOSIS.

The diagnosis of bronchogenic cancer is usually not difficult if the physician will consider the possibility of its existence. The fact remains that in most instances there is a delay in diagnosis of several valuable months. In this series, the average delay was 7.6 months before positive diagnosis. The average delay in diagnosis after the patient first sought medical consultation was 3.1 months. Less than 20 per cent of the patients had symptoms of two months or less.

The chest roentgenogram is the most valuable single means of obtaining an early presumptive diagnosis of bronchogenic cancer. In the present series, roentgenography led to a suspicion of bronchogenic carcinoma in 221 cases, or 97 per cent. Thus, the routine use of the chest X-ray in men over 40 years of age who are *not* having symptoms should lead to detection of bronchogenic carcinoma in many cases before the disease has progressed to a dangerous stage. In an

analysis of 51 cases of lung cancer with five-year postoperative survival, Overholt and Bougas[13] found that the disease was apprehended in all cases on the basis of a suspicious roentgenogram.

Blades[14] has shown that 70 per cent of asymtomatic patients with positive chest roentgenograms who have prompt thoracic exploration will have malignant pulmonic lesions with no lymphatic spread or metastases. Goldman and Freeman[15] emphasize the need for complete Roentgen study of the chest in every case in which symptoms of bronchial irritation are present, even for a few weeks. This should include, in addition to the usual postero-anterior views, the lateral, oblique, and inspiratory and expiratory films as well as fluoroscopy, which has been advocated by Garland.[16]

TABLE I.
SITES YIELDING POSITIVE RESULTS OF BRONCHOSCOPIC BIOPSY IN 92 CASES.

Right intermediate bronchus	18
Left lower lobe bronchus	16
Right lower lobe bronchus	14
Left main bronchus	11
Right upper lobe bronchus	11
Right main bronchus	11
Trachea	4
Left upper lobe bronchus	4
Right middle lobe bronchus	2
Carina	1
Total	92

Bronchography is of value in demonstrating bronchial obstruction in the segmental branches and the upper lobes. It frequently reveals associated bronchiectasis or pulmonary abscess.

Bronchoscopy should be performed in patients suspected of having bronchogenic carcinoma in order to establish the diagnosis by visualization of the lesion, forceps biopsy and bronchial aspiration and lavage for cytologic study. Bronchoscopy is also an aid in determining operability.

There were 1,112 bronchoscopic examinations for diagnostic purposes performed between 1943 and 1956. In this

group, there were 228 cases of bronchogenic carcinoma proved by biopsy, exploration or autopsy. A positive diagnosis was made by bronchoscopic biopsy in 92 (40.4 per cent) of the cases. The areas in the tracheobronchial tree which yielded a positive bronchoscopic biopsy are shown in Table I.

The types of abnormalities noted on bronchoscopy were the presence of an endobronchial reddish, fungating, papillomatous, easily bleeding mass or a polypoid, mulberry-like tumor. In some instances, an ulceration with rounded edges and a necrotic base was noted. In others, mural induration or bulging of the bronchus afforded a positive biopsy. It is in this latter group that there is the greatest possibility of error as the neoplasm is submucosal. The use of the bronchial telescope and flexible upper lobe forceps is an aid in the diagnosis and biopsy of tumors originating in the upper lobes.

The bronchoscopic demonstration of anatomico-pathologic changes indicative of bronchogenic carcinoma or its metastases, when correlated with the roentgenologic and clinical observations, comprises important suggestive evidence of malignancy in the absence of a positive biopsy report or negative cytologic report of bronchial aspirate. Of the 228 patients in the present series, 84 (36.8 per cent) had anatomico-pathologic changes which supported the clinical and roentgenographic diagnosis of bronchial cancer.

In the past decade, the collection of bronchial secretions by bronchoscopic aspiration and lavage for cytologic study has become a valuable diagnostic procedure. Clerf and Herbut[17] have done much to emphasize the importance of cytologic diagnosis in early lung cancer. They state that "the percentage of positive bronchoscopic biopsy bears a direct ratio to the percentage of inoperable cases". According to Gregg and associates[18] the utilization of endoscopically directed bronchial abrasion, irrigation, and aspiration of the mucosa result in higher percentages of positive diagnosis.

In 61 cases of proved primary cancer of the lung in the present series, cytologic examination indicated cancer in 29, or 47.5 per cent, which is 12.7 per cent of the entire series.

Bronchoscopic biopsy was positive in 92 cases (40.4 per cent); therefore, utilizing biopsy and cytologic diagnostic techniques, a positive diagnosis of lung cancer was made in 121 patients, or 53 per cent of the entire series. Bronchial aspiration with cytologic examination of secretions has been used roughly in the last half of this series; therefore, it seems certain that the percentage of positive diagnosis would have been much higher if the method had been in use in the earlier years.

Following a previous report[12] on this group, much higher percentages of positive diagnosis by cytologic methods have occurred, while the rate of positive diagnosis by biopsy has reduced. In the more recent group, a positive diagnosis of bronchogenic carcinoma was made by bronchoscopic biopsy in 25.9 per cent, and by cytologic examination of bronchoscopically recovered secretions in 55.5 per cent of cases; thus it was possible to prove bronchoscopically the existence of lung cancer in 81.4 per cent of the patients. It is noted that this figure is higher than the 53.1 per cent previously reported.[12]

The present technique of bronchial aspiration consists not only of collection of secretions from the suspicious lobe or segment of the tracheobronchial tree but also the vigorous lavage of such areas, utilizing abrasive application of suction tubes to the bronchial mucosa. The curved flexible upper lobe forceps and cannulae are useful in obtaining positive specimens from the upper lobes. The areas under study are washed with saline and immediately aspirated. Usually 20 cc. or more of saline is used for the lavage, divided into 10 cc. amounts. On occasion, lavage specimens have been collected from several segments of the lobe if the roentgenograms do not clearly show the apparent site of origin of the neoplasm. These specimens are studied separately by the pathologist, after the Papanicolaou technique of preparation and staining.

This examination involves a number of variables. The diagnosis of positive or negative secretions is directly proportional to the experience and technical analysis of the pathologist. It is difficult to differentiate normal and ab-

normal isolated tumor cells. Extreme care must be used in the examination, so that no small section containing neoplastic cells is missed. Generally speaking, we have used broncholavage and aspiration specimens in diagnostic study rather than sputum specimens because we have found, as pointed out by others,[19,20] that early in bronchogenic carcinoma the cough is nonproductive of cells; furthermore, sputum examination does not help to localize the carcinoma in the pulmonary bed.

SUMMARY AND CONCLUSIONS.

The incidence of bronchogenic carcinoma in the male population of the United States is showing a more rapid increase than that of any other malignant neoplasm.

The bronchoscopist should play an important role in the early diagnosis of lung cancer.

Bronchogenic carcinoma occurs primarily in white males in the fifth, sixth, and seventh decades of life. In this report, nearly 88 per cent of the cases were men.

Numerous atmospheric pollutants may be responsible for the causation of lung cancer. Certain industrial groups may be exposed to a number of well defined and highly potent respiratory carcinogens. Cigaret smoking may be a contributory factor, but may play a minor role in the etiology of bronchogenic carcinoma. In this series, 88.9 per cent of the patients smoked tobacco and 65.7 per cent of the total number were excessive smokers. Most of the patients were city dwellers and 65 per cent of the group had indoor occupations, while 35 per cent had worked predominantly outdoors.

There were 103 instances of squamous cell type carcinoma, 77 cases of undifferentiated carcinoma and 48 patients with adenocarcinoma. While only a small percentage of the epidermoid and undifferentiated carcinomas occurred in women, a more nearly equal relationship was found in adenocarcinoma.

The cardinal symptoms of bronchogenic carcinoma in order of frequency in this series were cough, lingering respiratory

symptoms following influenza, chest cold or pneumonia, chest discomfort or pain, dyspnea and hemoptysis. The average duration of symptoms from onset to positive diagnosis was 7.6 months. The average delay from the first medical consultation to positive diagnosis was 3.1 months.

The physical signs of bronchogenic carcinoma are those of any endobronchial obstruction in most instances.

The roentgenogram of the chest is the most valuable single means of obtaining a presumptive diagnosis of pulmonary cancer. In this series, 97 per cent of the patients were suspected by the roentgenologist as having bronchogenic carcinoma.

Bronchoscopy is of value in establishing the diagnosis of bronchogenic carcinoma by visualization and biopsy of the lesion, and by permitting broncholavage of selected areas of the pulmonary tree for cytologic examination. Bronchoscopy is also an aid in determining operability based on the presence or absence of anatomic-pathologic changes in the tracheobronchial tree typical of bronchogenic carcinoma. In this series, the diagnosis was made by bronchoscopic biopsy in 40.4 per cent of patients. In 84 patients, or 36.8 per cent, bronchoscopy demonstrated anatomico-pathologic changes in the tracheobronchial tree suggestive of bronchogenic carcinoma.

Cytologic study of bronchial aspirate specimens, after broncholavage, was done in 61 patients with the presence of malignant cells noted in 29 patients, or 47.5 per cent.

A high percentage of cytologic study positive for malignant cells in the bronchial aspirate is possible by abrasion of the bronchial wall with bronchoscopic forceps or aspirator followed by forceful washing of such areas with saline. In the later years of this series, cytologic diagnosis was positive in 55.5 per cent of cases, while forceps biopsy was positive in 25.9 per cent.

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765 Brotherhood Building.

CONDUCTIVE DEAFNESS DUE TO PYLE'S DISEASE.*

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Columbus, Ohio.

Conductive deafness associated with a normal tympanic membrane is generally attributed to otosclerosis; however, diseases other than otosclerosis cause conductive deafness and do not affect the appearance of the tympanic membrane. Among them are osteogenesis imperfecta, Paget's disease, and certain cranio-facial dysostoses.

In 1931 there was first described a condition which later became known as familial metaphyseal dysplasia, or Pyle's disease.[1] Since the original description of this particular bony dystrophy, a few additional cases have been reported.[2,3] The characteristics of this disease include a splayed or "Erlenmeyer flask" appearance of the long bones, especially the femora. These bony changes are best seen by roentgenographic methods. In familial metaphyseal dysplasia, the splayed conformation of the long bones is a primary condition and without known cause.

The present report concerns three unusual cases of Pyle's disease. They are unusual, because in addition to the changes in the skeletal bones previously described, these patients demonstrated a striking hyperostosis of the facial bones, cranium, and mandible. The cases are even more unusual because the patients gave deafness as the presenting symptom. None of the previously reported patients with Pyle's disease has been deaf.

REPORT OF CASES.

Case 1: K. G., an eight-year-old white boy (see Fig. 1), was seen at the Otology Clinic, University Hospitals, Ann Arbor, Mich., because of deafness, which was first noted when he was approximately two years of age. He had no history of middle ear disease, and his health had always been excellent. He was considered a bright child. A tonsillectomy

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Fig. 1. Case 1.

and adenoidectomy had been performed in an effort to relieve his deafness.

At the time of the preliminary examination, the child was obviously very hard of hearing. General physical examination was normal except for an appearance of hypertelorism, bossing of the frontal bones, and extremely prominent mastoid processes. The otologic physicial examination was negative, except for absence of the tonsils and adenoids and a moderately severe conductive deafness.

Barany chair testing elicited a normal response. The tympanic mem-

branes were normal in appearance and moved visibly when the patient swallowed. The Rinne test was strongly negative bilaterally. The patient heard a moderately loud spoken voice one foot from either ear, but did not hear a whispered voice. The audiogram is shown in Fig. 2. This illustrates excellent bone conduction and an average air conduction loss of 50 db bilaterally.

Complete laboratory examinations were within normal limits. The serological test for syphilis was negative.

Roentgenograms showing the entire bony skeleton were made. Most striking features from the otolaryngologic viewpoint were the great size of the mastoid bones caused by proliferation of cortical bone, and

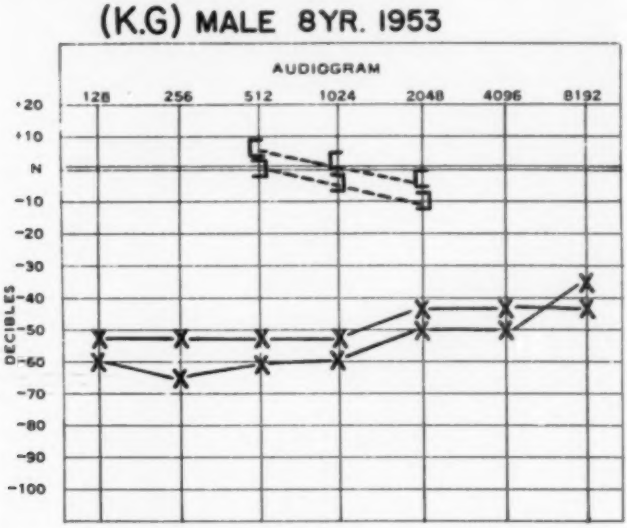

Fig. 2.

lack of pneumatization of the mastoid bones and paranasal sinuses (see Fig. 3). The radiologist commented particularly on the metaphyseal dysplasia represented by the splayed appearance of the long bones. The internal auditory meatuses were narrowed, and the foramina at the base of the skull appeared smaller than normal. A complete report of the radiographic findings will be published elsewhere.[4]

The Heredity Clinic found a strong familial tendency toward this disease. The maternal uncle, the mother, and the maternal grandfather were also deaf. In addition, they showed the same peculiar facies. In all four, the deafness started at an early age and became progressively worse. The patient's mother said that she had become deaf when she was about five years old and that her hearing was then stationary for several years. She had become increasingly deaf since

she was 12 years of age. Her father and one brother likewise had become hard-of-hearing as children and had gradually become more deaf as they grew older. Both are now dead. K. G. has shown no progression of his deafness in the past two years.

Case 2: J. V., a nine-year-old boy, is shown in Fig. 4. He has no known deaf relatives. He became hard of hearing at approximately five years of age. His deafness had not progressed in the last two years. His mentality has been considered normal. His past health has been good except for one attack of otitis media. This infection prompted an adenoidectomy and tonsillectomy.

Fig. 3. Case 1. Note proliferation of cortical bone and dense mastoid bone.

The tympanic membranes showed slight scarring, but were mobile when tested with the pneumatic otoscope and also moved when the patient swallowed.

There was bilateral peripheral facial paralysis. The patient also showed marked visual disturbances including optic atrophy and epiphora. The epiphora was attributed to bilateral osseous occlusion of the naso-lacrimal ducts. The optic atrophy was due to narrowing of the optic foramina by bony proliferation. Operative intervention at widening of the optic foramina was recommended by the Department of Neurosurgery, but was refused by the parents.

Functional testing of the ears showed a normal reaction in the Barany chair. There was a marked conductive type deafness, with some

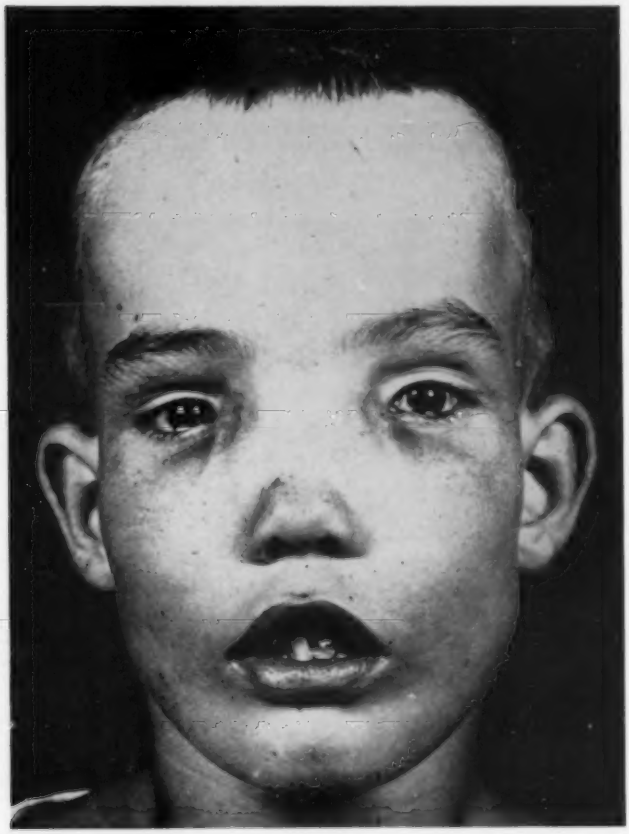

Fig. 4. Case 2. Note pooling of tears, expressionless face and hypertelorism.

neural loss superimposed. The Rinne test was strongly negative bilaterally. An audiogram is shown in Fig. 5.

Complete laboratory examinations were normal, and the serological test for syphilis was negative.

Roentgenograms were made of the entire bony skeleton and were interpreted as characteristic of Pyle's disease, or familial metaphyseal dysplasia. The lateral Roentgenogram of the skull is shown in Fig. 6.

Case 3: A 30-year-old white woman, the mother of the first patient, first noticed deafness when she was about four or five years old. Her deafness

did not progress until she reached puberty; then it became progressively worse. Her history was essentially negative. She recalled no middle ear infections.

A complete physical examination was negative. Routine laboratory tests were negative. Vestibular reactions were normal. The patient could hear a 512 cps. tuning fork only if it were struck a maximal blow, and could not hear any other fork. This patient wore a bone conduction hearing aid and claimed better results from it than from an air conduction hearing aid.

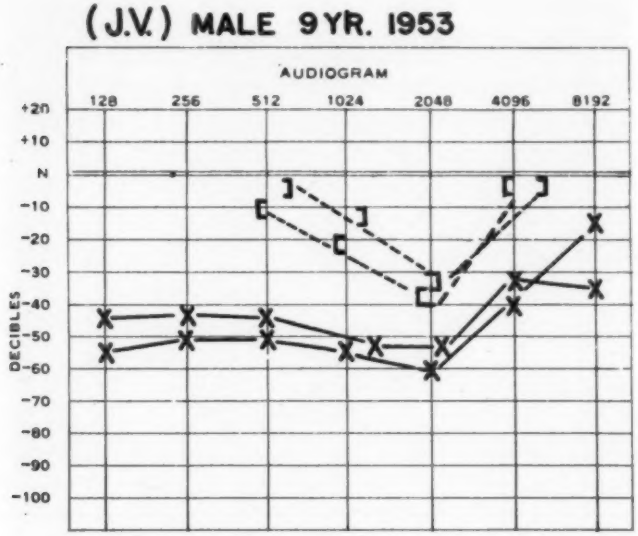

Fig. 5. Case 2.

Roentgenograms of the skull and long bones were reported by the radiologists as showing metaphyseal dysplasia and lack of pneumatization of the mastoid bones. This patient's facial appearance was very much like that of her son. She had a wide interpupillary distance, bossing of the frontal bones, and prominent mastoid processes.

DISCUSSION.

The mechanism producing deafness in these patients is unknown; however, it seems likely that the impedance element of the deafness resulted from fixation of the stapes in the oval window, or obliteration of the round window, or both.

In all three patients the tympanic membranes were essentially normal and moved upon swallowing. One of the children demonstrated a pure conductive deafness. The other child showed a deafness which was largely conductive, but also showed some loss of nerve function in the 1024 and 2048 frequencies.

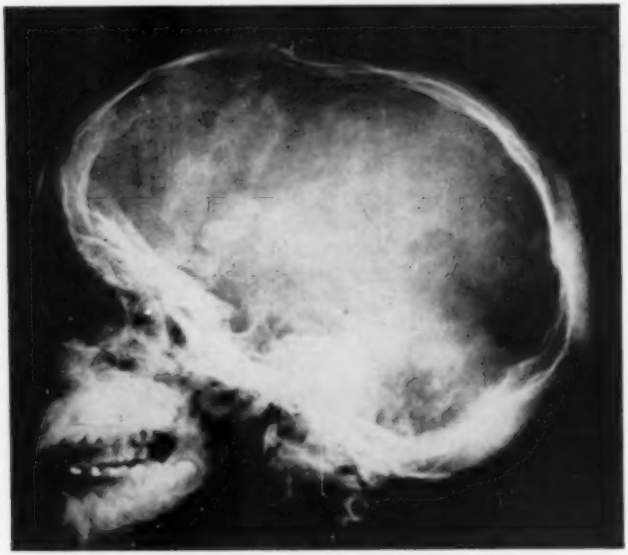

Fig. 6. Case 2.

In the woman (the mother of the first patient), the deafness was much more severe and showed a strong neural component. One may assume that in the mother there may have been encroachment by bony proliferation upon the cochlea itself, or upon the auditory nerve at the internal auditory meatus. This reasoning is supported by the appearance of the second patient, J. V., who demonstrated an osseous narrowing of the naso-lacrimal ducts and the optic foramina, as well as other cranial foramina.

In view of the severe nerve-type deafness in the adult pa-

tient, the prognosis for the hearing of the two children is uncertain. If the process of bony proliferation continues, they may become as deaf as the adult patient.

No therapy has been attempted in any of these patients except the fitting of hearing aids.

SUMMARY.

Three cases of deafness due to Pyle's disease have been presented. The cases differ from otosclerosis in the roentgenographic aspects of the temporal bones and long bones, and in the eventual severity of the deafness.

The changes in the skull and facial bones have not been associated with the previously reported cases of Pyle's disease, and deafness associated with this condition has never before been reported.

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The Department of Otolaryngology, Indiana University School of Medicine offers its annual Anatomical and Clinical Course in Otolaryngology March 25th to April 6th, 1957. Applicants should address The Post-Graduate Office, Indiana University Medical Center, Indianapolis 7, Ind.

DYSPHONIA AS A REACTION TO CIGARET SMOKE.

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and
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This is a report on a case who presented rather a unique manifestation of dysphonia, presumably on an hysterical basis:

Mrs. M. B., a 38-year-old white housewife, was first seen in the Otolaryngology Clinic in October, 1956, with the chief complaint of hoarseness upon exposure to cigaret smoke. She reported that no other type of smoke, such as that from burning leaves, coal, etc., affected her. She had never been a smoker and her husband was only a mild smoker. The only member of her immediate family who smoked at all was her father, who smoked heavily. She reported that she had never experienced hoarseness from any cause other than cigaret smoke, and that there was never an accumulation of post-nasal discharge upon her awakening in the morning. Her recovery from the periods of hoarseness seemed to be a function of the length of exposure; brief exposures were followed by almost immediate recovery, whereas lengthy exposures required several hours or overnight for the return of normal voice.

At examination, voice quality was pleasant and consistent with her age. Examination of the nose, nasopharynx, mouth, oropharynx, ears and neck was normal. Indirect laryngoscopy revealed the cords to be smooth, white, glistening, knife-sharp, and of normal mobility. General physical examination was not remarkable. Routine general screening tests were within normal limits. The patient was referred to the Speech Clinic for examination and treatment.

During this interview, the clinician ignited a cigaret in a small, closed room with the patient in proximity to the smoke. She was given material to read aloud while smoke was blown at her. As soon as a substantial quantity of smoke was in her vicinity, the voice was heard to drop sharply in pitch and assume a raspy, hoarse quality. The vocal cords during this period of hoarseness were examinted by indirect laryngoscopy and found to have remained normal in appearance. After an exposure of several minutes, the cigaret was extinguished, and the windows and doors to the room were opened. The return of the voice to normal was almost instantaneous. A similar episode approximately 20 minutes later, involving a pipe with tobacco, produced no change whatever in the quality of the voice. The cigaret experiment was repeated during a subsequent interview one week later with essentially the same results in terms of sudden onset of hoarseness.

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It was suggested to the patient that her condition could be based on either allergic or emotional factors. She was willing to be referred to an allergist for study, but would not consent to psychological evaluation. Her only concern was to discover a method for relieving the hoarseness when it occurred. In view of her attitude toward the problem, she was referred to an allergist who reported no reaction to a scratch test involving tobacco.

That dysphonia may be on an hysterical basis has been suggested repeatedly in the literature by various clinicians. Typically, this manifestation of an emotional problem has been treated successfully only through some form of psychotherapy; attempts to relieve the symptoms often result in the appearance of a new conversion outlet.

The patient was discharged after receiving suggestions in the use of a breathy type of phonation to be employed during the times that she was actually hoarse. It was explained to her that this type of phonation, which she grasped quickly, should help to prevent traumatic, pathological damage to the vocal cords. The only other suggestion was that she avoid cigaret smoke insofar as possible.

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INTERNATIONAL VOICE CONFERENCE.

Following the International Congress of Otolaryngology in Washington, D. C., next Spring, there will be an International Voice Conference (Laryngeal Research, Function and Therapy) in Chicago, Illinois, May 20-22, 1957. For Information address: Dr. Hans von Leden, 30 North Michigan, Chicago 2, Ill., U.S.A.

A METHOD OF RESTING THE VOCAL CORDS.

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In the treatment of conditions of the larynx which follow vocal abuse such as improper use of the singing or speaking voice, rest is a treatment prescribed. Rest of the vocal apparatus is indicated in such pathological conditions as laryngeal tuberculosis or laryngitis; also in those conditions which are the result of vocal abuse of long standing, *i.e.*, contact ulcers, contact granulomas, fiberous nodules, etc.*

A suggested treatment is based upon the observation of many otologists that individuals with an uncomplicated conductive hearing loss tend to speak softly. These individuals have the equivalent of a built-in pair of ear stoppers. Due to their conductive loss, when they are in noisy areas they do not have to raise their voices to hear their own conversations.

We can analyze this approach on a quantitative basis and estimate the theoretical results obtainable. If we place a person with normal hearing in a noisy office where the sound level is about 80 db, we find that in order to carry on conversation he must raise his voice to about 90 db level. Conversation carried on at this level for long periods of time tends to traumatize the vocal cords, especially if these are already irritated by some pathological condition.

The proposed treatment depends upon artificially producing a conductive hearing loss. Let us take the same normal hearing individual and by means of ear stoppers or earmuffs produce a conductive hearing loss of 20 db. If

*I am obligated to Dr. Simon Jesberg for limiting the application as shown in the first paragraph, and to Douglas Wheeler, Ph.D. for evaluating the acoustical values involved.

Note: Other than myself, I know of no one who has used this method of resting the vocal apparatus. Its future value will depend upon careful observations by the otolaryngologist; however, physicians with whom I have discussed the matter can see no reason why the procedure should be harmful.

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we now place this individual in the same surroundings where the background noise is approximately 80 db we will have the following situation: While the background noise is still 80 db, now by means of the ear stoppers we have eliminated 20 db. He now hears only 80 minus 20, or 60 db of this background noise. This is about the level of normal conversation, and consequently he does not raise his voice and does not add to the injury of the already traumatized vocal cords. The individual wearing the ear stoppers has no difficulty in hearing conversation since persons speaking to him tend to raise their voices above the level of the masking noise, *i.e.*, paracusis Willisii.

The question immediately arises: how does the individual who is not protected by ear stoppers hear under these conditions? From my own experience I find that the person protected by the ear stoppers tends to get close when he talks and probably tends to articulate more clearly; however the primary purpose of this arrangement is for the individual having the pathology or trauma to rest his vocal cords.

If these observations are correct, then it is conceivable that a method may now be available to avoid additional trauma to the vocal cords when conditions exist, and where rest of the larynx is indicated. It may also be a means whereby individuals such as salesmen or public speakers working in noisy areas, may be able to speak for longer periods of time without becoming hoarse and losing their voices.

This idea opens another, if somewhat speculative, approach to a problem in the field of noise, namely the controversy between those who feel that individuals working in noisy areas tend to be more tired at the end of the day's work than those working in quiet areas, and those who refute this idea. Are we dealing here with two separate and distinct problems? Are those who are tired and irritated the individuals who are required to make constant use of their voices, while unaffected individuals are those whose work is of only a mechanical nature?

SUMMARY.

Individuals with an uncomplicated conductive hearing loss do not tend to raise their voices in noisy areas as much as persons with normal hearing. By the use of ear stoppers or earmuffs in normal hearing individuals we can produce a condition which simulates a conductive loss. Since the individual who does not tend to raise his voice rests his larynx, this may be a means of treating conditions requiring rest of that organ.

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SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

CHRONIC SUPPURATION OF THE TEMPORAL BONE.

OPENERS: Marcus Diamant, Central County Hospital, Halmstad, Sweden—Anatomical Etiological Factors in Chronic Middle Ear Discharge.

Luzius Ruedi, Zurich, Switzerland—Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone.

Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany—Surgical Repair for Improvement of Hearing in Chronic Otitis Media.

DISCUSSERS: A. Tumarkin, Liverpool, England; Juan Manuel Tato, Buenos Aires, Argentina; T. E. Cawthorne, London, England; Fritz Zöllner, Freiburg, Germany.

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT.

OPENERS: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada.

Introduction:

Michele Arslan, Padua, Italy—The Upper Respiratory Tract.

Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.—The Lower Respiratory Tract.

DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

PAPILLOMA OF THE LARYNX.

OPENERS: Jo Ono, Tokyo, Japan—Etiology.

Plinio de Mattos Barretto, Faculty of Medicine, University of Sao Paulo, Brazil.

Diagnosis:

F. C. W. Capps, London, England—Therapy.

DISCUSSERS: C. A. Hamberger, Göteborg, Sweden; Pedro Hernandez Gonzalo, Havana, Cuba; Eelco Huizinga, Groningen, Netherlands; Albert von Riccabona, Vienna, Austria.

Anyone planning to attend the Congress and who has not yet registered should do so immediately in order to obtain hotel registration priority.

For more detailed information pertaining to the Sixth International Congress please communicate with the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

MOUNT SINAI HOSPITAL SPECIAL COURSE.

A special course in Rhinoplasty and Reconstructive Surgery of the Septum, following the Sixth International Congress of Otolaryngology, May 13, 1957, to May 18, 1957, will be given at the Mount Sinai Hospital, New York under the direction of Dr. Irving B. Goldman. This will be open to only foreign colleagues on a full scholarship basis.

An intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given July 13, 1957, to July 27, 1957, by Dr. Irving B. Goldman and staff at the Mount Sinai Hospital, New York in affiliation with Columbia University.

Candidates for either course should apply to Registrar for Post-graduate Medical Instruction, the Mount Sinai Hospital, Fifth Avenue and One-hundredth street, New York 29, New York.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill. ....
Vice-President: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Washington, D. C., May 4, 1957.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. LeRoy A. Schall, Boston, Mass.
First Vice-President: Dr. Henry M. Goodyear, Cincinnati, Ohio.
Second Vice-President: Dr. Robert E. Priest, Minneapolis, Minn.
Secretary: Dr. Harry P. Schenck, Philadelphia, Pa.
Treasurer: Dr. Fred W. Dixon, Cleveland, Ohio.
Meeting: Statler Hotel, Washington, D. C., May 3, 1957.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Percy Ireland, Toronto, Canada.
President-Elect: Dr. Lewis F. Morrison.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Washington, D. C., May, 1957.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

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Vice-Chairman: Dr. Kenneth L. Craft, Indianapolis, Ind.
Secretary: Dr. Hugh A. Kuhn, Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 13-19, 1957.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Clarence W. Engler, 2323 Prospect Ave., Cleveland, Ohio.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 21-23, 1958.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October 6-12, 1957.

THE AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. James Chessen, 1829 High St., Denver, Colo.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Illinois, October, 1956.
Annual Meeting: Palmer House, Chicago, Illinois, October, 1957.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLCGIC ALLERGY.

President: Dr. D. M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1957.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Irvin J. Fine, 506 New Brunswick Ave., Perth Amboy, N. J.
Secretary: Dr. William Schwartz, 224 Lexington Ave., Passaic, N. J.
Meetings: Quarterly.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracure, N. Y.
Secretary: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1957.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worchester, Mass.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Jose Gros, Havana, Cuba.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1958.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago 11, Ill.
Meeting: Statler Hotel, Washington, D. C., May 5 - 10, 1957.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Chevalier L. Jackson.
Vice-President: Dr. John J. O'Keefe.
Treasurer: Dr. Joseph P. Atkins.
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Historian: Dr. Herman B. Cohen.
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Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

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President: Dr. Raymond Kerwin, 310 So. Michigan Ave., Chicago, Ill.
Vice-President: Dr. Frank Wojniak, 6132 So. Kedzie Ave., Chicago, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

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Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

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Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Sherman B. Forbes, 706 Franklin Street, Tampa, Florida.
Vice-Chairman: Dr. Walter T. Hotchkiss, 541 Lincoln Road, Miami Beach, Florida.
Chairman-Elect: Dr. V. Eugene Holcombe, Medical Arts Building, Charleston, West Virginia.
Secretary: Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Virginia.

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President-Elect: Dr. Emanuel U. Wallerstein, Professional Building, Richmond, Virginia.
Vice-President: Dr. Calvin T. Burton, Medical Arts Building, Roanoke, Virginia.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Virginia.
Meeting: Roanoke, Virginia, December 6 and 7, 1957.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.
Annual Meeting: Greenbrier, White Sulphur Springs, W. Va., May 31st through June 1st.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

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Vice-President: Dr. A. J. McComiskey, 3420 Prytonia St., New Orleans, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: The Edgewater Gulf Hotel, Edgewater Park, Miss., May 17-18, 1957.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

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Vice-President: Dr. George E. Bradord, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting:

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Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

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Vice-President: Dr. Max M. Kulvin.
Secretary-Treasurer: Dr. James H. Mendel, Jr., 7241 Red Road, Miami 43, Florida.
Meeting quarterly (March, May, October and December), on the second Thursday of the month, 6:30 P.M. at Seven Seas Restaurant.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Ludwig A. Michael, 3707 Gaston Ave., Dallas, Tex.
Vice-President: Dr. Hal W. Maxwell.
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Chairman of Otolaryngology Section: Harold Boyd, M.D.
Secretary of Otolaryngology Section: Howard G. Gottschalk, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.
Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P. M. first Thursday of each month from September to June inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: H. Leroy Goss, M.D., 620 Cobb Bldg., Seattle 1, Washington.
Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah.
Meeting: April 7-11, 1957.

THE SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Col. Wendell A. Weiler.
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Time and place of meeting: October 1957, Palmer House, Chicago, Ill.

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Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

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Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

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President: Dr. G. M. T. Hazen, 208 Canada Bldg., Saskatoon, Sask.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: Banff Springs Hotel, Banff, Canada, June 17-19, 1957.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

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Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadephia 40, Pa., U. S. A.
Meeting: Sixth International Congress of Bronchoesophagology, Philadelphia, May 12-13, 1957.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

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Secretary:
Meeting: Lima, Peru, 1957.

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Secretary: Dr. Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon.

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Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1°, Lisboa.

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Vice-Presidente: Dr. Luis Suñe Medan.
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Vice-Secretario: Dr. A. Pinart.
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Second Secretary: Dr. Roberto Franco de Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filno. Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA
Havre 7—Desp. 62
Mexico 6, D. F.

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President: Dr. Máximo García Castañeda.
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THE NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY.
(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. J. Kuilman, Surinamestraat 19—Den Haag.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1—Amsterdam.
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Members: Dr. J. de Graaf, Dr. S. Muller, Dr. H. Navis.

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First Treasurer: Dr. Israel Scherman.
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THE SCOTTISH OTOLARYNGOLOGICAL SOCIETY.

President: E. A. M. Connal, 1 Royal Crescent, Glasgow C. 3, Scotland.
Secretary-Treasurer: Dr. J. F. Birrell, 14 Moray Place, Edinburgh.
Assistant Secretary: Dr. H. D. Brown Kelly, 11 Sandyford Place, Glasgow.

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